SUMMARY OF COMMENTS OF CREOSOTE COUNCIL II ON EPA'S DRAFT CREOSOTE HUMAN RISK CHARACTERIZATION, DRAFT PRELIMINARY RISK ASSESSMENT, AND ASSOCIATED DRAFT REREGISTRATION ELIGIBILITY DECISION (RED) SCIENCE CHAPTERS

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INTRODUCTION

Creosote Council II (the "Council") is a FIFRA § 3(c)(2)(B) joint data development group, which has sponsored and submitted all of the studies and data required by EPA in connection with the reregistration of creosote. The Council appreciates the opportunity to comment on the advance draft preliminary risk assessment and associated draft science chapters, which the Antimicrobials Division provided to the Council in January 2003.

As discussed below and in the appended expert reports, **the current draft risk assessment and science chapters are riddled with serious errors, such as numerous assertions and conclusions based on inaccurate, inappropriate, or incomplete information, and major flaws in scientific approach**. These include, but are not limited to, the following:

- EPA continues to treat creosote as an agricultural use, crop protection pesticide instead of an industrial pesticide. This results in the application of the wrong paradigm for creosote assessment by EPA;

- EPA has expanded its evaluation of creosote to include creosote-treated wood products but did not require registrants to generate or submit exposure or hazard data on treated wood products. In the absence of such data, EPA completed its hazard and risk assessments with inappropriate data from sources unrelatable to creosote and creosote-treated wood.

- EPA frequently states or suggests that creosote registrants have failed to provide data to support certain reregistration efforts. The registrants have provided all data required by EPA except for one study that is currently underway.

- The draft toxicology characterization of creosote misreads or ignores creosote toxicity studies submitted by the Council.

- The draft human health risk assessment relies upon a single surrogate substance for cancer assessment and a single surrogate substance for noncancer effects. This approach has been criticized (for coal tar-derived mixtures like creosote) in the past by the National Research Council, and is susceptible to criticism today.

- The draft environmental risk assessment fails to incorporate existing literature on ecotoxicity and environmental fate of creosote, and fails to use existing validated models for creosote environmental fate in freshwater, marine and land environments.

The Council strongly urges EPA not to release the preliminary risk assessment or draft science chapters for public comment until after thoroughly considering all of the Council's comments, and making the necessary revisions.

Following below are general comments on each of the draft chapters provided to the Council. In addition to these general comments, the Council is submitting detailed scientific critiques prepared by four experts: Dr. Robert Tardiff prepared comments on EPA's human health risk assessment; Dr. Mark Bookbinder prepared comments on the agency review of the "Assessment of Potential Creosote Inhalation and Dermal Exposure Associated with Pressure-Treatment of Wood with Creosote" (the worker exposure study); Dr. Kenneth Brooks prepared comments on ecotoxicity, environmental fate and the ecological risk assessment of creosote; and Mr. Stephen Smith prepared a report on environmental issues relating to creosote, specifically the draft chapters on Product Use Profile and Environmental Modeling.

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I. USE PROFILE

As the Agency notes in the Regulatory History for creosote, this material has been successfully used as a wood preservative pesticide for a long time, a time that predates both the US EPA and the Department of Agriculture. With regard to the draft Creosote Use Profile, the appropriate CAS Registry Number to be used for coal tar creosote is 8001-58-9, and that is the registry number that should be used for the wood preservative creosote.

The list of creosote registrants in the EPA draft is inaccurate: Osmose, Inc., now known as Osmose Utilities Services, Inc., is not a basic manufacturer of creosote, but is a formulator. It is the understanding of the Council that Carbolineum Wood Preservation Company and IBC Manufacturing Company no longer hold creosote registrations. Reilly Industries, Inc. has been acquired by KMG-Bernuth, Inc., and Western Tar Products Corporation is now known as Railworks Wood Products, Inc. A listing of the Council believes to be the current end-use creosote FIFRA registrations appears below.

Creosote, which is defined by American Wood-Preservers' Association (AWPA) Standards P1/P13 and P2, is a terrestrial non-food crop, restricted-use pesticide. It is not an agricultural use pesticide. The pesticidal uses of creosote are limited to wood preservation, and the overwhelming majority of that is for pressure-treatment of wood. More than 99.99% of pesticide creosote sales are into the pressure treating market. Both P1/P13 and P2 creosotes, which are coal tar creosotes, are used in wood pressure treating, and are the active ingredient in the registered end-use products. Pressure-treating and non-pressure treating wood preservation uses of creosote are outlined in the attached report of Mr. Stephen T. Smith (Appendix I). The formulated use product containing creosote as an active ingredient is, as stated above, Osmoplast. Osmoplast is a paste (or grease) containing 45.62% creosote that is applied by brush, trowel or pump for remedial treatment to existing wood structures.

US EPA CREOSOTE PESTICIDE REGISTRATION NUMBERS

Coopers Creek Chemical Corp.	Registration Number
Coopersote Creosote Oil.	363-15
Black Creosote Coal Tar Solution	363-14
KMG-Bernuth, Inc.	
Creosote Oil 24CB	61483-7
Creosote Coal Tar Solution P2	61483-8
Creosote Oil P1	61483-9
Creosote P3	61483-10
Creosote Oil P1/P13	61483-11*
Creosote Oil Coal Tar Solution	61483-12*
*Formerly Reilly Industries, Inc. creosote registrations, 1456-18 and 1456-20	
Koppers Industries, Inc.	
Coal Tar Creosote (Pressure Applications)	61468-1
Creosote Solution (Pressure Applications)	61468-3
Coal Tar Creosote (General Applications)	61468-5
Creosote (Manufacturing Use)	61468-6
Railworks Wood Products (Formerly Western Tar Products, Inc,.)	
Creosote	2458-1-73408
Creosote Solution	2458-4-73408
Rutgers VFT (KMG-Bernuth, agent)	
KMG-B Creosote P1	61470-1
KMG-B Creosote P2	61470-2
Trenton Sales, Inc. **	54774-1
Osmose Utilities Services, Inc.***	
Osmoplast	75341-7

** Not a member of Creosote Council II but a contributor to the P1/P13 North American Creosote Composite Test Material.

*** Formulator; not a member of Creosote Council II

II. PRODUCT CHEMISTRY

Creosote is a complex mixture of variable composition derived from the distillation of crude coal tar. Like any distillate stream mixture, creosote is most usefully and reliably defined in terms of its physical/chemical properties which remain relatively constant, as opposed to its molecular composition. To identify creosote, the Council (and the wood treating industry) adhere to the use of the American Wood-Preservers' Association (AWPA) standards for P1/P13 and P2 coal tar creosote. These standards provide unambiguous criteria for what can be considered "creosote" for use in wood preservation.

More specifically, the AWPA standards for creosote define the pesticide material that is registered with EPA and the material which registrants are supporting with data submissions to EPA. The commercial pesticide creosote products of each member of the Council meet AWPA creosote standards, as does the creosote composite test materials used to actually generate creosote reregistration data. By assuring that the composite test materials and each registrants' creosote products meet the same standard, the AWPA standards, the creosote registrants can warrant that their products are equivalent to what was actually evaluated in data development work, and that what was evaluated is equivalent to the creosote that is in commerce. For these reasons, the testing methods for compliance with AWPA P1/P13 and P2 creosote standards are the methods most appropriate for certification of creosote as an active ingredient in a pesticide.

The Council conducted EPA FIFRA Product Chemistry Guideline chemistry testing for Preliminary Analysis (Guideline 62-1), Physical/Chemical Properties (Guideline 63) and AWPA Standard Specification (for identity), as well as long-term stability analysis and corrosion on the Composite Test Materials and each contributor's commercial products, 60 study volumes, in all. The work was done at a leading independent laboratory and the studies were conducted and reported in accord with FIFRA GLP requirements. Following completion of the work and submission of the reports to EPA, the agency conducted a quality assurance audit of the testing facility (Research Triangle Institute, Research Triangle Park, N.C.) and of the underlying study data. The EPA auditors had no adverse findings to report for their site inspection or for the condition and completeness of the study data.

The Council feels that in the draft RED chapter on Product Chemistry EPA places too great an emphasis on general reference texts, historical information, publications from the open literature or environmental reports of little relevance for information about creosote product chemistry. Clearly, the most relevant and well-controlled data set on creosote chemistry is the data set submitted by the Council, and this should be the source of information EPA uses to inform itself about creosote chemistry.

The Agency's grouping of creosote components by chemical family (Tables 1-3) in the draft chapter may be useful for data analysis, but the Council is concerned that

listing of "Relative Percentage" in each of the table could give a misimpression about the amount of each component actually present in creosote. The "Relative Percentage" as shown in the tables is the ratio of each component in that EPA grouping. It is not the percentage of each component in creosote. It is not clear from the draft chapter what utility there may be in listing components of creosote in this manner. Table 4, for instance, lists the actual concentration of each identified component in P1/P13 and P2 creosotes. The note appearing at the end of Table 4 correctly indicates that individual creosote component quantitation was performed with calibration standards generated on four marker components. However, it should be noted that identification of each component was accomplished through the use of two separate mass spectral libraries: the NIH/EPA/MSDC Mass Spectral Data Base and the Registry of Mass Spectral Data. In addition, authentic certified standards were used for exact compound identification for 57 of the 144 components or isomers of P1/P13 and P2 creosotes. The statement in note 1 concerning water solubility does not relate to GC/MS analysis and appears to be misplaced.

The section of the draft Product Chemistry chapter dealing with storage stability and corrosion (pages 14 and 15 of the draft report) erroneously states that data were submitted for three fractions of creosote: P1, P2, and P1/P13. There is no AWPA standard for P1 creosote; no data on P1 creosote was submitted to EPA by the Council.

III. RESIDUE CHEMISTRY

In this brief chapter, EPA affirms that creosote does not have agricultural base uses and should not have indirect food contact use. The agency agrees that there is minimal cause for concern at this time about creosote residue and direct food contact, food contact surfaces, trophic transfer or biomagnification. EPA does state that in the event that further research shows a high degree of bioaccumulation or biomagnification due to creosote, additional data may be required to address these points. The Council agrees with this assessment, and urges EPA to review the report prepared by Dr. Kenneth Brooks (Appendix II) in which the extant data on bioaccumulation and biomagnification of creosote and its components are discussed in detail.

IV. EPIDEMIOLOGY AND INCIDENTS

The Council is concerned that the human health incident reports for creosote have not been authenticated in any way by EPA, but nevertheless are presented in the draft chapter on human health effects of creosote. There is no verification of the allegations accompanying the case reports. The published reports are predominantly from text books, i.e., secondary sources, which themselves may be of a somewhat current publication date but are citing very old primary source material.

One example of this is the report of eye injury cited in Grant's Toxicology of the Eye, 1986. The description of corneal injury and the duration of involvement originated from German case reports dating from 1913 as well as reports from 1938, 1943 and 1955.

These observations are completely different from eye effects reported by the Council in 1993 animal eye testing of creosote. In that testing, which used direct instillation of undiluted liquid creosote into the rabbit eye – an animal model considered to be more sensitive than the human eye - P1/P13 creosote was mildly irritating to the eyes and P2 creosote is moderately irritating to the eyes. Animal eyes were evaluated for damage to the cornea, the iris and the conjunctiva. Evaluations were made using an ascending scale of 0 to 110. No corneal or iris changes were reported for either creosote at any time point (all scores "0"). Only conjunctival irritation was produced by direct introduction of liquid creosote into rabbit eyes: the irritation was completely reversible in 100% of the cases. The maximum irritation score for P1/P13 creosote occurred at 24 hours post-dosing and was 7.0 out of a possible 110.0. The maximum irritation score for P2 creosote occurred at 1 hours post-dosing and was also 7.0 out of a possible 110.0. The difference in reports of the potential for creosote to produce ocular toxicity may derive from differences in creosote composition (over 80 years) or differences in creosote composition between Europe and the US. It is known from work completed in 1963, that early testing of creosote produced a more severe eye response than what was observed in 1993. The number of animals used in the 1963 work was less than the later work, but the overall protocol and eye response scoring scheme was the same for both studies. This suggests that differences in creosote composition may have occurred over a number of years that manifest as differences in creosote toxicity. There is also the possibility of misidentification of the original contaminant reported in the 1913 German literature. Furthermore, in many instances, several texts site the same primary source leaving the impression that there are a greater number of reports than actually exist.

Because of the uncertainty associated with incident and case reports, the Council would like EPA to indicate in the chapter on human health effects of creosote the level of confidence that should properly be associated with these reports.

V. REVIEW OF WORKER EXPOSURE STUDY

In the draft review of the Council submission, "Assessment of Potential Creosote Inhalation and Dermal Exposure Associated with Pressure-Treatment of Wood with Creosote" (the worker exposure study), EPA summarized the study design and study results and offered several criticisms of the study report. These criticisms are addressed in detail in the attached report of Dr. Mark Bookbinder, the Study Director, as Appendix III.

The view of EPA that inhalation exposure to creosote should be expressed as "total creosote" instead of components or Coal Tar Pitch Volatiles (CTPV) is inappropriate since airborne exposure to vapors will consist of volatilized components of creosote separately but in proportion to their individual vapor pressure and percent composition in creosote, and exposure to aerosolized creosote, i.e., droplets, are captured (and reported) as part of the CTPV. In his comments, Dr. Bookbinder offers a method for the exposure determination that EPA suggests, and from his examples it appears that such an approach results in airborne creosote exposure estimates that are lower than those

reported by the Council in the worker exposure study using conventional methods for assessment of airborne exposure to mixtures.

The Agency also questioned why the Council report did not state the amount of creosote active ingredient used at treating sites as a metric of worker exposure (dose). As pointed out by Dr. Bookbinder and contrary to statements by EPA in the draft chapters, the quantities of creosote applied to each charge of wood are known and reported in the Council report. Table XII of the Council report provides this information. EPA is correct in stating that this information was not used in calculating a metric for worker exposure to the wood preservatives. This is because there is no useful way to quantitatively extrapolate worker exposure from the amount of pesticide used at a site. EPA is also correct is stating that the amount of preservative pesticide to which treating workers are exposed is a very small fraction of the amount charged into the treating cylinders. Since only wood and not workers are exposed to the preservatives inside of a treating cylinder during pressure treatment, there is no useful way to quantitatively extrapolate worker exposure from the amount of pesticide used at a site. The purpose of the Council study was to obtain measurements of pesticide exposure at the level of the workers' skin and in their breathing zones as they engaged in normal wood treating work activities over the course of several days. An analysis of those measurements did yield quantitative data on pesticide exposures to specific workers that could be associated with specific job activities and suggested that there are differences in exposure levels among test sites and job activities.

EPA incorrectly criticizes the study on the basis that insufficient field recovery data, and that blanks were presented in the study and correction calculations were not expressed. In his comments, Dr. Bookbinder explains that adequate field and blank control samples were included in the creosote worker exposure study. In addition to field spiking controls, both pre-field testing of target analytes and concurrent laboratory spiking of collection matrices were conducted for this study. The results of pre-field spiking were presented in Tables IX, page 101 of the study report. Laboratory spiking results are presented in Tables XVIII through XVIII, report pages 120-125. (Field spiking results are presented in Tables XV and XVI, pages 114-119.) The results indicated that pre-field recoveries of metal-spiked samples were adequate for the proposed field samples. The number and types of field controls employed in this study follows Series 875 Occupational and Residential Exposure Test Guidelines Group C – Exposure Monitoring Test Guidelines. Moreover, this plan for field spikes was described in the study protocol reviewed and approved by EPA and the Joint Agencies prior to study initiation.

The concerns of EPA regarding recovery values for some of the field fortification spike controls are also addressed by Dr. Bookbinder in his comments. He explains that loss of some portion of the highly volatile components of creosote from sample collection media was anticipated and that steps to account for this were in place during the conduct of the study and in data analysis.

Dr. Bookbinder has also included clarification and examples of calculations performed on study data to improve the transparency of data transformations.

As a last point, EPA indicated in the Risk Characterization draft chapter that there are "issues" with the analytical portion of the study. The agency did not identify any such issues, so that the Council can respond.

VI. TOXICOLOGY

The draft chapters sometimes state or imply that creosote registrants have failed to submit particular studies which EPA possibly may need or like to have in connection with certain assessments. For example, page 1 of the Toxicology chapter states that "[t]here are no current Agency guideline neurotoxicity studies available for creosote." The draft chapter fails to note, however, that no neurotoxicity studies on creosote ever have been required by EPA. Similar statements appearing in the risk characterization draft chapter (and in other draft chapters) give the impression that the creosote registrants have not done what was required under the Reregistration Standard and Data Call-In Notice. Page 3 states that "data gaps exist". This impression is incorrect, and all such misleading statements should be deleted from the draft chapters. While it may be true that the registrants have not conducted studies which, in hindsight, EPA might like to have in its possession, it is also true that in no instance has the Council failed to submit a study that has been required by EPA. (The lone exception to this is the Protective Clothing Permeability Study which is presently underway at Midwest Research Institute.)

The assertions in the draft chapter of creosote-induced systemic toxicity are overstated. Creosote does not produce significant eye irritation. As stated above, undiluted P1/P13 and P2 creosotes were tested in rabbit eyes for irritation. The results indicated that P1/P13 creosote is mildly irritating, and that P2 creosote is moderately irritating, to the eyes. Evaluations were made using a standard ascending scale of 0 to 110. No corneal or iris changes were reported for either creosote at any time point (all scores "0"). Only conjunctival irritation was produced by direct introduction of liquid creosote into rabbit eyes; the irritation was completely reversible in 100% of the cases. EPA classifies pesticides with this type of eye irritation potential into Pesticide Category IV, a category that requires the Signal Word "CAUTION" to appear on the product label but does not require additional precautionary label language.

The assertion that creosote is neurotoxic appears in several sections of the draft Toxicology chapter, each in the absence of actual neurotoxicity data. The discussion of beechwood creosote on page 1 of the draft chapter is entirely irrelevant to coal tar creosote toxicology, as is the parenthetical mention of naphthalene as a neurotoxin. The European Union Chemicals Branch risk assessment of naphthalene, the most current summary of naphthalene toxicity information, does not cite any neurotoxicity findings for naphthalene. The ATSDR Toxicology Profile for naphthalene says that reports of brain damage in humans following fatal or near fatal naphthalene ingestion is due to cerebral edema secondary to naphthalene-induced hemolysis or kernicterus secondary to liver failure. This is not evidence of neurotoxicity any more than a skull fracture is considered

evidence of neurotoxicity. The observation of lethargy in high-dose animal studies of naphthalene were not supported by histopathological findings of neurotoxicity. In the eight repeated-dose studies of creosote, conducted by dermal and inhalation exposures, there were no reports of daily pharmacotoxic observations that would support a finding of neurotoxicity and no evidence of nerve tissue damage from gross and microscopic examination of the animal tissue.

Cardiotoxicity and pulmonary changes are cited as toxicological effects of concern by EPA. For the subchronic inhalations studies rats were exposed in whole-body inhalation chambers to PI/P13 or P2 creosote for 6 hours/day 5days/week for 13 weeks. The concentrations were 106, 59 and 5.4 mg/m^3 for P1/P13 creosote, and 102, 48 and 4.7 mg/m^3 for P2 creosote, respectively. The No Observed Adverse Effect (NOAEL) for these studies was the low exposure concentration, about 5 mg/m^3, meaning that exposure to a creosote aerosol at this level was without treatment-related effects. To lend perspective to this exposure level, 5mg/m^3 is the occupational exposure limit for mineral oils. The mid- and high-dose exposure levels of creosote were approximately ten and twenty-fold above the low-dose, and animals in these test groups were exposed to a visible mist of airborne creosote. Such exposure to those levels of creosote (vapor and particulate) produced body weight loss or weight gain suppression in the top two exposure groups and staining of the animals' respiratory tract and lungs immediately after the 90-day exposure period. Creosote deposition in the nose and upper respiratory tract produced acute and chronic inflammation which decreased along the tract toward the lungs. According to the study pathologist, the lung changes were limited to pigment deposition characterized as trace level in severity and there were no lung inflammatory changes or lung lesions of any type that could be associated with the pigment. Two of 20 high-dose and one of 20 mid-dose animal exposed to P1/P13 showed signs of myocardial degeneration on the right side of their heart. The Thyroid changes (follicular cell hypertrophy) were noted in creosote-exposed animals; this condition was reversible in the animals exposed to P1/P13 creosote.

Six weeks after the creosote exposures were terminated, a subgroup of animals that had been exposed during the testing period were sacrificed and examined microscopically. Trace levels of pigment remained in the lungs and nose along with cyst formation in the nose. Thyroid changes disappeared in the P1/P13 animals but remained in P2 animals. No changes indicative of heart damage were observed in any animal following the recovery period. At the end of the exposure period and at the end of the recovery period animals were evaluated by a veterinary ophthalmologist for signs of eye damage due to airborne creosote. No test material related eye changes were recorded.

The agency has misread the developmental toxicity studies submitted by the Council. Contrary to the statements in the draft Toxicity Chapter, there was significant maternal toxicity at dose levels below the doses that produced embryo or uterine effects in the P1/P13 rabbit and P2 rat studies. This means that there is not reason to consider creosote as a developmental toxicant and that the developing fetus is not a target for creosote toxicity. In reviewing the P1/P13 rat developmental toxicity the agency failed to consider that the incidence of eye malformations they coiste as evidence for creosote

acting as a developmental toxicant fell within the historical control incidence for that malformation for the performing laboratory and the study director considered the eye findings as not creosote related.

Finally, the agency makes a statement indicating that the reproductive toxicity study submitted by the Council has deficiencies but did not explain what those deficiencies might be and how, if at all, they affect the conclusions of the study.

VII. HUMAN EXPOSURE

The Council's comments on this draft chapter are contained in the comments on the draft human risk characterization chapter.

VIII. HUMAN RISK CHARACTERIZATION

EPA's draft human risk characterization is flawed in that it is overly conservative and fails to utilize data more appropriate for assessment of creosote risk than the creosote surrogates chosen by EPA, naphthalene and benz(a)pyrene. The Agency assessment overestimates exposure as well as overstating toxicity hazard. A critique of the EPA risk characterization for creosote is presented by Dr. Robert Tardiff in Appendix IV.

EPA erroneously states on page 2 of the draft risk characterization that the registrants failed to submit retrospective epidemiology data. Creosote registrants were not required to submit epidemiology data, but were required to submit data on the feasibility of conducting an industry epidemiology study. This was done in a June 20, 1993 submission to OPPTS. Along the same lines, page 4 of the draft risk characterization chapter states that "no chemical-specific data for post-application exposure was submitted to the Agency". This statement is repeated on page 6 and is followed by the statement, "post-application exposures to residents ... could not be assessed due to lack of data." As noted elsewhere in the Council comments, such statements implying that creosote registrants have failed to submit *required* studies that EPA needs to complete certain assessments are false and misleading, and should be deleted.

The Council takes exception to the use of benz(a)pyrene as an indicator of carcinogenic risk and naphthalene as an indicator of noncancer occupational risk for creosote. Details of the objections are provided by Dr. Tardiff in his report. The Council believes that in general the carcinogenic/mutagenic effect of specific PAHs is not a reliable indicator of the effect of PAHs in a complex mix like creosote. In other words, no one indicator molecule can adequately serve as a surrogate for the toxicity of a complex mixture like creosote. Broadly speaking, the reasons for this are that the hydrocarbon components of creosote, planar fused ring compounds (PAH), vary considerably in their biological activity and, importantly, in their metabolism from species to species. Since PAH's are indirect-acting or promutagens, meaning that genotoxicity is expressed following metabolic conversion of the PAH to an active

species, metabolism is a critical factor in the genotoxicity and carcinogenicity of PAH's. Just as metabolic transformation of PAH's is necessary to covert the PAH into an active mutagen or carcinogen, there is information to suggest that metabolic addition of a substituent group, almost always a methyl group, on a PAH impacts on PAH biologic activity. There are numerous examples of the alkylation of the PAH (with a methyl group) both enhancing and eliminating PAH tumorigenicity and mutagenicity. One illuminating work evaluated the carcinogenic effect of methyl-, ethyl-, and propyl substitutions on chrysene.
Methylchrysene was a more potent lung carcinogen that chrysene, but ethyl- and propylchrysene were less potent. Methylation has been shown to transform inactive PAH's to active and to nonactive carcinogenic PAH's. Bay region methylation (methyl addition at specific site) of dimethylbenzanthracene, a potent mutagen and carcinogen, completely blocks mutagenic and carcinogenic activity (Amin, et al, 1991).

The use of individual PAH components of a mixture, or any individual component, as a surrogate for the mix is fraught with possibilities for mistake. There is no validated method for tracking or otherwise characterizing the fate of mixtures of variable composition through the use of an indicator molecule. The US National Research Council Report from the Board on Environmental Studies and Toxicology stated in their report on Complex Mixtures – Methods For In Vivo Testing that indicator molecules have been used to assess mixtures based on the behavior of a constituent on the assumption that the toxicological behavior of the constituent is representative of the mixture. In judging this approach, the Committee cited the example of BaP. The committee reviewed independent epidemiological research from six investigators which collectively indicate that the BaP content of two complex mixtures, one of which was coal tar pitch volatiles, failed to correlate with the end point (lung cancer) associated with that mixture. The National Research Council report concludes that "BaP is at best is a crude indicator of the carcinogenic potential of complex mixtures"(NRC, 1988).

In fact, direct testing of the role BaP can play in pulmonary carcinogenesis caused by a complex PAH mixture (soot and carbon black) has demonstrated an inverse relationship between BaP and tumor formation. The relationship was also inversely dose-related, meaning that the greater the amount of BaP administered with a tar product, the lower the incidence of lung tumors (Dasenbrock et al, 1996). Culp et al (1998) and Gaylor (1988) evaluated the tumorigenicity of two coal tar mixtures and compared those to BaP in lifetime rodent studies. The authors concluded that although the tumor potencies of the coal tar mixtures were comparable in terms of target organ and tumor production efficiency, BaP failed to trigger a similar qualitative and quantitative tumor response and specifically failed at the most sensitive tumor site (lung). They concluded that the current value for BaP cancer potency is incorrect and that the use of BaP-based toxicity equivalency factors " to estimate lung cancer risks associated with mixtures of PAH's appears inappropriate". Several areas of investigations offer insight or possible explanation to these observations: mechanistic studies as described in Section 3 below provide convincing evidence of the interactions among PAH components of complex coal tar derived mixtures, and on a pharmacokinetic level, the work of Ingram (1995), for instance, showed that the bioavailability of dermally applied BaP as determined by

epidermal protein and DNA BaP binding, varied by as much as 14-fold as a result of animal grooming and viscosity of the BaP-containing oil.

Accordingly, BaP content is not a reliable indicator of the potential carcinogenicity of a complex hydrocarbon mixtures and should not be a consideration in the classification of creosote.

The Council believes that it is inappropriate to base the occupational creosote hazard assessment solely on naphthalene when more relevant creosote data exist. The agency should consider the subchronic inhalation studies completed on P1/P13 and P2 creosotes as a basis for creosote hazard characterization. In any case, it is incomprehensible to apply a derivation of a residential exposure standard to occupational exposure conditions (for a noncancer endpoint) when an occupational exposure standard exists, as it does for naphthalene. The Council wishes to point out that although naphthalene was detected in air sampling at each worker site, in no instance did the naphthalene air concentration approach the established occupational exposure threshold.

EPA's use of the Pesticide Handlers Exposure Database (PHED) and the Chemical Manufacturers Association Exposure database (CMA) is irrelevant for creosote wood treating applications. Although these databases were used for 10 of the 28 exposure scenarios considered by EPA, the nonpressure-treating uses of creosote (those to which the PHED and CMA databases were applied) constitute less than 0.1% of creosote pesticide usage.

IX. ENVIRONMENTAL EXPOSURE AND MODELING

X. ENVIRONMENTAL FATE

XI. ECOLOGICAL EFFECTS AND ENVIRONMENTAL RISK CHARACTERIZATION

These three draft chapters are addressed by Mr. Steve Smith and Dr. Kenneth Brooks in Appendices I and II. Brief summaries of the Smith and Brooks comments are presented below.

The Council believes that the Agency's approach to environmental fate assessment for creosote is superficial and does not approach the current state of science for what could be done to assess creosote environmental fate. Dr. Brooks, with access to the same information in the open and technical literature as EPA, identifies a number of relevant publications and even creosote-specific fate models that have been overlooked by EPA. Furthermore, the Council strongly disagrees with statements throughout the draft Environmental Fate Chapter and the draft Ecological Effects and Environmental Risk Chapter suggesting or stating that creosote registrants have failed to submit studies EPA needs to assess ecotoxicological or environmental hazard or risk. For example, the first sentence of the Data Summary for Environmental Fate Assessment, page 2 of the draft chapter, says, "Registrant studies have not been submitted to the Agency and

specific guideline requirements have not been fulfilled for creosote". This statement is contradicted by the May 4, 1995 letter from EPA OPPTS to the Council which states as follows:

> The Agency has evaluated the status of the subject requirements (creosote ecological fate and ecological effects data requirements) in light of concerns about environmental fate tracking; the nature and variability of creosote components; how traditional pesticide data requirements might apply; and the viability of a risk assessment derived from these types of studies. In consideration of these factors, the Agency will allow the creosote registrants the option of satisfying the environmental fate and ecological effects data requirements by conducting a literature search in lieu of conducting additional eco-toxicity or environmental fate studies. Ample data are available in the open literature on the exposure, degradation pathways and toxicity to nontarget organisms in both freshwater and estuarine marine environments for each component of the composites, P2 and P1/P13, such that no further testing should be necessary.

Emphasis added.

In place of ecological and environmental fate testing, the Council submitted a literature search to EPA. Virtually every section of the Ecological Effects draft chapter ends with a statement saying that the registrants have not submitted any studies addressing the effects discussed in that particular section of the chapter. These statements appear despite EPA's admission on page 23 of the draft chapter that the registrants were given an opportunity to conduct a literature search in lieu of testing and that "there are no data gaps" ecotoxicity.

EPA has used an inappropriate model for estimating environmental exposure to creosote. Stephen Smith outlines in his report the issues of concern the Council has with the use of the GENEEC model for estimating creosote transport through environmental compartments. Chief among these concerns is that the GENEEC model is designed for estimating the fate of agricultural chemicals applied directly to soil or crops by broadcast methods of application. This is not even remotely relevant to the use of creosote in pressure treating or the use of creosote pressure-treated wood. Furthermore, the assumptions about creosote loss from pressure-treated wood are unrealistically high and the degradation of creosote components are unrealistically low. The Council hopes that after reviewing the work of Dr. Brooks it will be abundantly clear that appropriate models do exist for describing the environmental fate of creosote and creosote-treated wood products.

The Council is fortunate to have Dr. Brooks comments on these drafts. He is well respected in the field of ecotoxicology and perhaps the leading expert in ecotoxicology and environmental fate of treated wood. His comments are best summarized by citing the conclusion to his view of the environmental fate portion of the draft chapters. Brooks states that

This entire section needs to be rewritten with more emphasis on traditional science and intellectual rigor. The literature review is incomplete at best and selective at worst. EPA has not properly focused its attention on the use of creosote treated products. Instead, despite the availability of numerous peer reviewed documents describing the environmental response to creosote, EPA has relied on laboratory studies using whole creosote oil, which is not characteristic of the suite of PAH lost from the product, on information from inappropriate historical industrial practices and hydrocarbon spills – none of which are indicative of the environmental response to the use of properly produced creosote treated wood products. Information specific to the use of creosote treated railway ties and to the environmental response to creosote treated wood used in bridge construction in freshwater and marine structures has been made available to EPA, but the agency has chosen to ignore this information. Instead, the agency has relied on studies published before year 2000. In closing, this section is woefully missing well supported interpretations of the literature or defensible conclusions. Where conclusions are provided, they are unsupported by relevant citations and in several instances EPA's conclusions are easily rebutted by the literature.

Dr. Brooks makes similar observations about ecological effects descriptions and risk characterization. In each instance he identifies appropriate literature EPA could have considered in constructing their chapters and forming their assessments of the environmental effects of creosote and creosote-treated wood products. Key literature discussed by Dr. Brooks will be supplied to EPA under separate cover. Any other references cited by Dr. Brooks will be made available to EPA upon request.

CONCLUSION

The Council is disappointed with the quality of the draft preliminary risk assessment and science chapters. The drafts reflect insufficient consideration of all of the available pertinent data, unwarranted assumptions, and insufficient coordination within EPA. The Antimicrobials Division should review the Council's comments, including the appended expert reports, and make all necessary revisions to the drafts prior to releasing them for public comment.

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APPENDICES

I Environmental issues review of USEPA draft preliminary risk assessment on c creosote.
Stephen T. Smith, P.E., AquAeter, Inc., Helena, MT.

II Comments regarding the Environmental Protection Agency's draft preliminary risk assessment on creosote.
Kenneth M. Brooks, Ph.D., Aquatic Environmental Sciences, Port Townsend, WA.

III Response to EPA comments concerning the creosote pressure-treatment worker exposure study included in the creosote human risk characterization preliminary risk assessment documents.
Mark G. Bookbinder, Ph.D., Germantown, MD.

IV Critique of USEPA's creosote human risk characterization.
Robert G. Tardiff, Ph.D., The Sapphire Group, Bethesda, MD.